



DEPARTMENT OF THE NAVY
NAVY EXPERIMENTAL DIVING UNIT
PANAMA CITY, FLORIDA 32407-5001

IN REPLY REFER TO:

NAVSEA Task 88-18A

DTIC FILE COPY

AD-A219 560

TIC
LECTE
R07 1990

Dcs D

NAVY EXPERIMENTAL DIVING UNIT

REPORT NO. 1-90

FIELD MANAGEMENT OF ACCIDENTAL HYPOTHERMIA
DURING DIVING

LCDR JOHN A. STERBA, MC, USNR

JANUARY 1990

DISTRIBUTION STATEMENT A: Approved for public release;
distribution is unlimited

Submitted:

Reviewed:

Approved:

John A. Sterba

J.A. STERBA
LDCR, MC, USNR
Research Medical Officer
Cold Water Studies
Project Manager

H.J.C. Schwartz

H.J.C. SCHWARTZ
CAPT, MC, USN
Senior Medical Officer

James E. Halwachs

JAMES E. HALWACHS
CDR, USN
Commanding Officer

H.L. Pruitt

H.L. PRUITT
LDCR, USN
Senior Projects Officer

J.B. McDonnell

J.B. McDONELL
LDCR, USN
Executive Officer

90 03 05 026

SECURITY CLASSIFICATION OF THIS PAGE

REPORT DOCUMENTATION PAGE				
1a. REPORT SECURITY CLASSIFICATION UNCLASSIFIED		1b. RESTRICTIVE MARKINGS		
2a. SECURITY CLASSIFICATION AUTHORITY		3. DISTRIBUTION/AVAILABILITY OF REPORT DISTRIBUTION STATEMENT A: Approved for public release; distribution is unlimited.		
2b. DECLASSIFICATION/DOWNGRADING SCHEDULE				
4. PERFORMING ORGANIZATION REPORT NUMBER(S) NEDU Report No. 1-90		5. MONITORING ORGANIZATION REPORT NUMBER(S)		
6a. NAME OF PERFORMING ORGANIZ. Navy Experimental Diving Unit	6b. OFFICE SYMBOL (If applicable)	7a. NAME OF MONITORING ORGANIZATION		
6c. ADDRESS (City, State, and ZIP Code) Panama City, FL 32407-5001		7b. ADDRESS (City, State, and ZIP Code)		
8a. NAME OF FUNDING/SPONSORING ORGANIZATION Naval Sea Systems Command	6b. OFFICE SYMBOL (If applicable) 00C	9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER		
8c. ADDRESS (City, State, and ZIP Code) Washington, D.C. 20362-5101		10. SOURCE OF FUNDING NUMBERS PROGRAM ELEMENT NO.	PROJECT NO.	TASK NO. 88-18A WORK UNIT ACCESSION NO.
11. TITLE (Include Security Classification) (U) Field Management of Accidental Hypothermia During Diving				
12. PERSONAL AUTHOR(S) STERBA, J. A.				
13a. TYPE OF REPORT FINAL	13b. TIME COVERED FROM _____ TO _____	14. DATE OF REPORT (Year,Month,Day) JANUARY 1990	15. PAGE COUNT 29	
16. SUPPLEMENTARY NOTATION				
17. COSATI CODES FIELD GROUP SUB-GROUP		18. SUBJECT TERMS (Continue on reverse if necessary and identify by block number) hypothermia, cold, therapy, diving, thermal protection		
19. ABSTRACT (Continue on reverse if necessary and identify by block number) Hypothermia of both the extremities and the body core continues to be a major problem in cold water diving. Presently, extremity hypothermia of the hands limits cold water diving exposure more so than body core hypothermia. Painful or numb fingers decrease dexterity, safety, work capacity, and increase the risk of developing nonfreezing cold injury (NFCI), which is reviewed. Levels of body core hypothermia, based on rectal core temperature, are mild (35°-32°), moderate (32°-28°), and severe hypothermia (below 28°C). As it relates to cold water diving, the pathophysiology of immersion and mild to severe hypothermia is reviewed including thermoregulation and effects on organ systems. The diving response, composed of bradycardia and peripheral vasoconstriction, has been shown in man to not prolong breath-holding time, or influence (CONTINUED)				
20. DISTRIBUTION/AVAILABILITY OF ABSTRACT <input type="checkbox"/> UNCLASSIFIED/UNLIMITED <input type="checkbox"/> SAME AS RPT. <input type="checkbox"/> DTIC USERS		21. ABSTRACT SECURITY CLASSIFICATION		
22a. NAME OF RESPONSIBLE INDIVIDUAL	22b. TELEPHONE (Include Area Code)	22c. OFFICE SYMBOL		

19. (Continued)

alveolar gas exchange, such as oxygen consumption. Survival from cold-water near-drowning may be more dependent on hypothermia than previously recognized. The predisposing factors leading to hypothermia during diving operations are discussed. The determination of body core temperature from various sites including new radio pill telemetry is reviewed. The levels of hypothermia by physical exam findings are reviewed in detail. The field treatment of hypothermia is discussed with attention to a review of the literature, recent research, and first aid management guidelines. The prevention of hypothermia during diving operations and current diving medicine research in the active thermal protection of free-swimming divers is reviewed. A comprehensive review of the literature provides 127 references.

CONTENTS

	Page No.
I. INTRODUCTION.....	1
A. EXTREMITY AND CORE HYPOTHERMIA.....	1
B. PATHOPHYSIOLOGY OF HYPOTHERMIA DURING DIVING.....	2
C. PREDISPOSING FACTORS LEADING TO HYPOTHERMIA.....	4
II. METHODS AND RESULTS.....	5
A. DETERMINING BODY CORE TEMPERATURE.....	5
B. CLINICAL ASSESSMENT OF THE HYPOTHERMIC DIVER.....	6
C. FIELD TREATMENT OF HYPOTHERMIA, A REVIEW OF THE LITERATURE.....	9
D. FIELD TREATMENT OF HYPOTHERMIA, CURRENT RESEARCH.....	10
III. DISCUSSION AND CONCLUSIONS.....	12
A. FIELD TREATMENT OF HYPOTHERMIA, FIRST AID MANAGEMENT.....	12
B. PREVENTION OF HYPOTHERMIA DURING DIVING.....	15
C. RESEARCH IN ACTIVE THERMAL PROTECTION FOR DIVING.....	15
REFERENCES.....	17

Accession For	
NTIS - CRASI	<input checked="" type="checkbox"/>
DTIC TAB	<input type="checkbox"/>
Unannounced	<input type="checkbox"/>
Justification	
By	
Distribution	
Availability Codes	
Dist	Special
A-1	

I. INTRODUCTION

A. EXTREMITY AND CORE HYPOTHERMIA

1. Extremity Hypothermia

Hypothermia continues to be a major problem in cold water diving. With thermal conductivity of water 26 times greater than air (1), hypothermia of the fingers and toes, called extremity hypothermia, is a frequent occurrence during cold water diving. The limits of thermal protection are becoming more dependent on avoiding extremity hypothermia due to intolerable hand and foot pain and the loss of hand dexterity (2). In extreme cold water, close to the freezing point, the finger tip temperature may drop to the pain threshold of 10° to 12°C in less than 30 mins if the diver in a dry suit is not generating sufficient heat by swimming or vigorous underwater work (3-5). This occurs hours before the core temperature would fall to hypothermic levels. Going beyond finger pain, into numbness (8° to 10°C), increases the risk of inducing non-freezing cold injury, NFCI (1). In fact, some divers have noted that their hands developed an unusual warm sensation subsequent to mild pain. Their digit temperatures of only $6-8^{\circ}\text{C}$ were within the numbness range for most divers (3,5). The mechanism of NFCI is unclear, but prolonged cold exposure and restriction of digit blood flow may permanently alter the normal vascular response to tissue rewarming and subsequent cold exposure (1,6). Following nonfreezing cold injury, the rewarming process is accompanied by swelling, redness, pain and itching, which is similar to the hyperemic response during rewarming frostbitten tissue. The treatment of prolonged cold exposure leading to numbness would be the same as for frostbite; immersion in $40-42^{\circ}\text{C}$ ($104-108^{\circ}\text{F}$) water for quick rewarming to limit tissue damage from the cold (7,8). Narcotic analgesia may be needed.

If NFCI has occurred, upon subsequent exposure to cold water, the diver will experience intense cold and pain very quickly. This is possibly due to early vasoconstriction in the digit blood vessels (1,6). The duration of cold exposure that will develop NFCI is not known. With slow cooling of the hands, the diver may never realize his hands have become numb.

Our laboratory uses the following termination criteria to prevent non-freezing cold injury during extreme cold water dives: any digit, finger or toe, to 8°C (46.4°F) for 30 mins, or any digit to 6°C (42.8°F), at any time (9). After approximately 20 man-dives with divers reaching this termination criteria, no diver has experienced non-freezing cold injury. However, four divers, with no prior history of non-freezing cold injury, noted either pins and needles (paresthesia) and decreased sensation (hypesthesia) lasting hours following termination due to the above criteria. Based only on anecdotal reports from military cold water diving units, we would estimate that 60 mins of digit numbness would probably cause nonfreezing cold injury in most divers. There appears to be great intersubject variability in susceptibility to NFCI. The prevention of this disabling problem, which can restrict diving in cold water, is being investigated by scientists who are now monitoring digit temperature during experimental and open-ocean dives in extreme cold water (3,5,9). Safe exposure limits for cold water diving can then be determined for dives using various factors including varying levels of underwater exercise,

water temperature, diving suit garment, breathing gas composition and body type.

2. Core Hypothermia

Clinical hypothermia is defined as a body core temperature, measured rectally, below 35°C (95°F) (10-13). Many authors have subdivided hypothermia into mild hypothermia (32- 35°C), moderate hypothermia (28-32°C) and severe hypothermia (below 28°C). However, rectal core temperature is frequently not available in the field. In the absence of core temperature the levels of hypothermia are correlated in this review by clinical signs, symptoms, and physical exam. This practical approach will help to direct which patients are only mildly hypothermic and may rewarm in the field, passively with appropriate support. Moderate and severe hypothermia require active rewarming by invasive methods, which should be attempted in a hospital setting.

Following cold exposure, the continual decline in body core temperature during the initial rewarming period is defined as body core afterdrop. The mechanism is from both on-going conductive cooling as well as convective cooling from peripheral cold blood returning to the body core (14).

B. PATHOPHYSIOLOGY OF HYPOTHERMIA DURING DIVING

In diving, there is a strong peripheral receptor input from cold skin receptors to the thermoregulatory center, the hypothalamus. Despite a normal core temperature, the diver may feel very uncomfortable with shivering and may have useless hands from extremity hypothermia. This "distraction effect" of feeling cold, shivering and having cold hands can compromise diver safety or the successful completion of a task requiring unimpaired cognitive and hand dexterity function (15).

By a central redistribution of approximately 700 ml of blood from immersion (16), which may be enhanced by peripheral vasoconstriction from very cold water, there is a brisk increase in urination (diuresis) (17-18). This high clearance of mostly free water can reduce plasma volume by 17% (19) leading to both reduced blood volume (hypovolemia) and dehydration following a dive. This will reduce ability of the diver to rewarm adequately, to safely perform duties topside or ashore following a dive, and will reduce his defense against hypothermia during repetitive dives. The effect during immersion to centrally displace blood, also supports the circulation of a hypothermic diver. After surfacing, loss of this "G-suit" effect of water hydrostatically compressing capacitance vessels in the legs becomes important in the handling of hypothermic divers and will be discussed below.

The complex physiological changes occurring with hypothermia affect every organ system. It is easiest to discuss these changes first with mild hypothermia, more commonly seen in diving, and second with moderate and severe hypothermia, seen more with drowning or severe exposure.

1. Mild Hypothermia

Immersion in cold water increases ventilation, causing respiratory alkalosis, and cerebral vasoconstriction leading to confusion and impaired

judgment (20-22). Both peripheral and central thermoreceptors cause a progression of thermogenic responses starting with an uncomfortable increase in muscle tone, called non-shivering thermogenesis (23). Later, suppressible shivering occurs and is soon replaced with nonsuppressible shivering. This can prevent the diver from completing useful work underwater, from helping himself or his buddy, or from climbing aboard ship without topside assistance. During deep diving with Helium-Oxygen (HeO_2), non-heated inspiratory gas, with very high thermoconductivity at depths greater than 100 m, can cause an insidious hypothermia without any warning signs to the diver (24-25). With the hot water suit surrounding the diver, peripheral receptors can override central thermoreceptors, preventing any discomfort or shivering. Loss of hot water to a saturation diver or deep surface supplied HeO_2 diver, is a true diving emergency and hypothermia should be anticipated.

2. Moderate and Severe Hypothermia

Diuresis continues due to anti-diuretic hormone (ADH) suppression, atrial natriuretic factor (ANF) secretion (26), and reduced renal tubular metabolism due to cold (27-28). The failure of cellular membrane metabolism causes intracellular volume to expand with further loss of plasma volume, compounding hypovolemia (13). The increase in hematocrit reflects the hemoconcentration and increase in blood viscosity (13). Changes in electrolyte and acid base balance are well reviewed (13), however, little can be done in the pre-hospital setting to alter these changes. With rewarming, especially from rapid onset hypothermia, these electrolyte and acid base changes self-correct (13). The circulatory system is compromised by intense vasoconstriction and reduced myocardial conduction and contractility. With cardiac output falling, due to bradycardia and reduced stroke volume, tissue metabolism becomes more anerobic. Metabolic acidosis is increased from reduced liver function, and lactate from shivering (13). With decreases in myocardial conduction, all EKG intervals tend to be prolonged. The P waves are diminished in amplitude, T waves are inverted and the pathognomonic elevated R-T segment, called the "J" wave or Osborne wave is frequently seen (29). The physiological mechanism of the "J" wave is unclear, and has been interpreted as a current of injury (30), delayed ventricular depolarization (31), or early repolarization (32). The appearance of a "J" wave is an ominous sign of moderate hypothermia (29), but should not direct one's therapy either in the field or in the intensive care unit. The critical progression of bradycardia, to increased atrial and then ventricular ectopy should warn the health care provider. Unless rewarming is proceeding well, asystole or ventricular fibrillation may soon develop, which are the terminal arrhythmias observed in severe hypothermia (13,33-35).

3. Cold-Water Near-Drowning

In a diving accident with cold-water near-drowning, severe hypothermia develops. In 1977, it was publicized that the mammalian dive reflex or diving response was responsible for survival during the accidental hypothermia caused by cold-water near-drowning (36-39). The diving response is a central redistribution of oxygenated blood at the expense of the periphery. It is characterized by both vasoconstriction and bradycardia and is induced by breath-holding and cold stimulus to the face. Seen in diving mammals, such as seals and whales, it also exists in man and has been extensively studied, especially the bradycardia response (40-47). Whether the diving response in

man could be of any physiological consequence by conserving oxygen and prolonging man's breath-holding time had yet to be investigated.

In 1979, it was first reported that when inducing the diving response with cold water submersion (20°C) the breath-holding time was greatly reduced in man (48). This was explained as exteroceptive cold stimulation possibly overriding the dive reflex (48). These results were later corroborated by Hayward et al. (49) who also observed shortened breath-holding times in colder water, 0°C . In 1985, it was further explained that this shortening of submersed breath-holding time by an increase in both metabolic rate as well as respiratory drive, overriding any physiological benefit of the diving response (50). Next, it was investigated whether breath-holding time could be enhanced by inducing the diving response but also avoiding the respiratory stimulus of whole body submersion in cold water (51). A marked diving response could be elicited by breath-holding and cooling only the face (52-56). This situation is similar to diving animals who have abundant insulation, except about the face. Diver-subjects were selected based on prescreening for the strongest diving response bradycardia from a large population of divers. In these studies, the diving response was potentiated by adding mild exercise (46,57) and breath-holding at less than maximal lung volumes (45,58-59). Despite a strongly induced reproducible diving response, demonstrated by reductions in heart rate, cardiac output and peripheral perfusion, there were no differences in breath-holding times, alveolar gas exchange or breaking point alveolar O_2 or CO_2 tensions (51). Even potentiating the diving response by exercise did not affect breath-holding times compared to control breath-holds with thermoneutral water face immersion (51). Very interestingly, if the face was continuously exposed to cold water, similar to a cold-water drowning, the diving response was abolished by probable cold adaptation (51).

These results determined in conscious, breath-holding man (51) do not support the theory that the diving response is physiologically responsible for survival from cold-water near-drowning. With the victim of cold-water near-drowning becoming unconscious, developing hypothermia and possibly liquid breathing with prolonged submersion, the diving response may physiologically contribute to survival. However, hypothermia is a more probable cause for remarkable recovery from cold-water near-drowning. Based on these studies, the facemask of an unconscious diver should not be removed underwater in attempts to induce the diving response.

C. PREDISPOSING FACTORS LEADING TO HYPOTHERMIA

The following factors are frequently noted during cold water diving operations, and may contribute to hypothermia (60-62).

1. Dehydration from repetitive diving, excess alcohol and caffeine consumption leading to diuresis, aversion to drinking from sea sickness, inadequate fresh water during Arctic/Antarctic diving operations, and elevated insensible water loss during strenuous outdoor activity without regular replenishment of fluids with sugar/electrolyte rehydration drinks.

2. Inadequate rewarming during repetitive cold water diving operations.

3. Diving after tending for prolonged periods leading to hypothermia before diving.

4. Dry suit undergarments that are wet due to inadequate drying from perspiration and/or dry suit leaks, cause up to a 88% reduction in insulation (63-64).

5. Profuse sweating during strenuous dives, reducing insulation of the undergarment, and then remaining at rest for prolonged decompression stops.

6. Inadequate thermal insulation: wet suit instead of dry suit, undergarment selection too thin, too compressible or of poor insulation when wet (63-64), inadequate thermal insulation of the head.

7. Neglecting to heat inspired HeO_2 below 300 feet (91 m) (25).

8. Poor physical conditioning, lack of cold acclimatization, poor physical health due to chronic disease or acute illness, alcohol consumption.

II. METHODS AND RESULTS

A DETERMINING BODY CORE TEMPERATURE

Determining the body core temperature in the field (e.g., aboard a dive boat, in a helicopter) is difficult with having to remove wet or dry suits to use flexible rectal thermistors. The radio frequency temperature pill developed in the early 1970's was used in human experimentation and in the evaluation of life support systems (65-66). However due to low demand, these pills were never marketed. Recently a new temperature pill (CorTemp, Human Technologies, Inc., St. Petersburg, FL) using the near-field-magnetic-link principle of telemetry was developed for in-hospital monitoring of patient temperature. At the Navy Experimental Diving Unit, the CorTemp unit antenna was modified to measure gastrointestinal (G.I.) temperature of a diver either dry or immersed. Eight divers swallowed these CorTemp pills during experimental induction of hypothermia and the evaluation of field rewarming techniques. Unfortunately, results were disappointing with 35 of 41 pills proven to be very inaccurate compared to two thermistors calibrated by National Bureau of Standards (Sterba, unpublished data). In addition, the data were unreliably recorded and there were frequent difficulties transferring the data into the personal computer for display purposes. These problems reportedly are, however, being corrected by Human Technologies, Inc.

There are criticisms of relying on a swallowed pill thermistor to determine the G.I. core temperature (67). The pill temperature would be temporarily influenced by cold or hot fluid that was swallowed with the pill. Also, hypothermic victims may not have a protective gag reflex, and should not be given fluids by mouth in order to swallow a temperature pill. Since the pill travels throughout the G.I. tract, temperatures may vary with location, for example being higher near the liver, a highly metabolic organ. Despite these concerns, using real-time telemetry data from a temperature pill might prove to be effective during rewarming in the field situation with a pill in the upper G.I. or placed deep within the rectum. Additional research is needed to prove the value of using temperature pills.

Although difficult to accomplish, rectal temperature has been used to monitor body core temperature in the field. Mercury low registering thermometers and standard clinic-type digital electronic thermometers cannot be inserted beyond 5-7 cm and will give falsely low temperatures from cold dependent blood returning via the venous plexus surrounding the distal rectum. This would adversely affect rewarming strategies. The rectal temperature probe should therefore be flexible (Yellow Spring Instruments (YSI), Yellow Springs, Ohio, model 700 series rectal probe, digital display and model 400 series, analog display.) The rectal probe must be inserted at least 15 cm from the anus to avoid the return of cold venous blood from the legs and secured with tape. The monitor must also have a scale well into the hypothermia range, to 21°C (70°F). A flexible rectal thermometer with a small radio frequency amplifier has been developed by the Canadian Defense and Civil Institute of Environmental Medicine (DCIEM), Downsview, Ontario. To date, this unit has not been marketed or tested under field conditions.

Obviously oral, axillary, and forehead skin temperature are both much lower than rectal temperature and are measured very unreliably outdoors in the cold. Tympanic membrane temperature reflects brain temperature, but no one has ever proven that it reflects the temperature of the thermoregulatory center, the hypothalamus. Tympanic membrane temperature also is influenced by the scalp and external ear temperature, plus it is very uncomfortably measured, making it impractical for monitoring in the field. Esophageal temperature, known to represent cardiac temperature (68-69) is an ideal temperature for environmental thermoregulation studies (70-71). However, it is not practical to attempt naso-esophageal insertion of such a probe in the field. Although rectal temperature lags behind esophageal temperature to monitor rapid changes in core temperature (68), deep insertion and taping a rectal thermister in place is presently the best method for core temperature monitoring in the field.

B. CLINICAL ASSESSMENT OF THE HYPOTHERMIC DIVER

It is exceptionally rare to have hypothermia worse than mild hypothermia during diving operations, unless there has been a drowning with prolonged recovery. Rectal core temperature monitoring takes considerable effort, e.g., removing diving gear. A rapid initial assessment of the hypothermic diver depends upon a close examination of mental status, peripheral pulse, and presence or absence of shivering. It is important to remember that the shivering response is maximum around 35°C rectal body core temperature (11,13), the upper limit of mild hypothermia. If there is an absence of shivering, the diver's overall condition will tell if he is sufficiently warm or dangerously dropping below 35°C . By closely observing the diver immediately following the dive and conducting a physical examination outlined below, a clinical determination of the degree of hypothermia can be made, even without a rectal temperature. These physical findings are based on direct observation of hypothermic patients with adequate core temperature monitoring (11,13,28,34,72-79).

1. Vital Signs

a. Rectal temperature. Mild hypothermia ($32-35^{\circ}\text{C}$), moderate hypothermia ($28-32^{\circ}\text{C}$), severe hypothermia (below 28°C).

b. Heart rate. Mild hypothermia: mild bradycardia is common when there is no shivering, but also tachycardia during episodes of shivering and with hypotension. Moderate hypothermia: pulse very weak with bradycardia more severe, irregular heart rate common with increased atrial ectopy and ventricular escape. Severe hypothermia: profound bradycardia, atrial and ventricular ectopy is worse, or pulseless from ventricular fibrillation or asystole.

c. Blood pressure. Mild hypothermia: blood pressure varies, may be hypertensive especially during intense shivering but frequently hypotensive. Attempt blood pressure monitoring in between episodes of shivering, else there will be a false elevation. Moderate and severe hypothermia: increasing bradycardia, severe ectopy, cardiac depression, and severe hypotension develop. Remember, with a detectable radial pulse, systolic blood pressure is at least above 80 mmHg; a femoral pulse, above 70 mmHg, and a carotid pulse, above 60 mmHg (80).

d. Respiratory rate. Mild hypothermia: usually elevated, and may be labored especially with intense shivering. Moderate to severe hypothermia: respiratory rate declines or is absent, can become very labored with bronchorrhea and lastly, pulmonary edema.

2. General Condition

With mild hypothermia, there is a range of shivering from intense to an insidious decline of shivering accompanied by sometimes an altered sense of body warmth. Victims succumbing to hypothermia have disrobed in the cold due to this dysesthesia of warmth. Confusion, stumbling gait, intense fatigue or sleepiness are common. The speech can be slurred from a cold face and central nervous system impairment. In moderate and severe hypothermia, the patient is not shivering, and may appear to be dying or be dead.

a. Mental Status. In mild hypothermia, judgment is frequently impaired, orientation is usually well preserved, but short-term memory is very reduced with a shortened attention span. The affect is very labile and the patient may be agitated to lethargic, along with either a high anxiety or depressed mood being common. With moderate and severe hypothermia, there is increasing obtundation and unresponsiveness to pain.

b. Skin. In mild hypothermia, skin color may be pale from vasoconstriction, but frequently the face and hands are cyanotic with venous congestion caused by dry suit hood squeeze and wrist cuff squeeze. Exposure to high wind chill and direct exposure to water temperatures below 10°C will induce peripheral vasodilation and skin erythema. In moderate to severe hypothermia, the skin becomes very pale and cyanotic, with capillary refill not being able to be determined.

c. Ears. With mild hypothermia pale, waxy color indicates intense vasoconstriction. Tympanic membranes can be very erythemic from cold exposure and may be misdiagnosed as barotrauma or mild middle ear squeeze (Teeds Class 1) (81). There are no remarkable findings with more severe hypothermia, except cyanosis of the external ear.

d. Eyes. From mild to severe hypothermia, the eyes increase in lackluster appearance, begin to lose reactivity in moderate hypothermia and are fixed, at mid-point to full dilation in severe hypothermia.

e. Throat. Frothy sputum from cold induced bronchorrhea is not seen in mild hypothermia, but can be common in moderate and severe hypothermia. During deep diving using helium with unheated inspiratory gas, bronchorrhea can develop quickly and may severely obstruct the airway of an exercising diver with normal body core temperature or mild hypothermia. The pink froth of pulmonary edema is a very late sign, seen in severe hypothermia.

f. Chest/Lungs. In mild hypothermia, assess the lungs by auscultation and percussion in between the severe episodes of shivering. The lungs should be clear. Rhonchi from upper airway obstruction from bronchorrhea is progressively observed from moderate to severe hypothermia. Rales due to frank pulmonary edema is a late sign in severe hypothermia.

g. Heart/EKG. As discussed above with assessing pulse, mild hypothermia is accompanied by a deepening bradycardia, separated by a more rapid heart rate with intense shivering. In moderate to severe hypothermia, there is a progression of increased atrial to increased ventricular ectopy, ending with asystole directly, or via ventricular fibrillation. Ventricular fibrillation spontaneously occurs and is refractory to defibrillation below 28°C (28). This defines the critical upper limit of severe hypothermia. The Osborne or "J"-wave, which is a conspicuous upward deflection at the R to ST wave junction, is often seen in mild to severe hypothermia, and may persist after adequate rewarming (30). Shivering artifact on the EKG must not be misinterpreted as ventricular fibrillation. With shivering in periodic episodes, continuous monitoring will allow reasonable EKG traces between bouts of shivering. We have had good success with using Lead II monitoring during underwater shivering if two leads are placed superiorly and inferiorly on the sternum to avoid chest muscle artifact and the third lead still be placed on the left mid-axillary line (Sterba, unpublished observation).

h. Musculoskeletal/Neurological. Emphasizing again, shivering is maximum at a rectal core temperature of 35°C, the upper limit of mild hypothermia. In between bouts of intense shivering the muscle tone and reflexes will be elevated in mild hypothermia, but will quickly become depressed in moderate to severe hypothermia. From 35°C to 32°C, shivering begins to decrease in intensity and frequency which can be dangerously misinterpreted as the patient not being that cold.

i. Abdomen/Urogenital. With the intense shivering and increased muscle tone of mild hypothermia, it is very difficult to initiate the relaxation phase of urination and there may be very painful bladder distension. Following cold water diving with mild hypothermia, urine volumes can exceed 800 ml, so be prepared! Following urination, anxiety and overall malaise from hypothermia is greatly reduced. This will greatly assist the health care provider to better evaluate the mental status and physical exam of the hypothermia victim.

C. FIELD TREATMENT OF HYPOTHERMIA, A REVIEW OF THE LITERATURE

Controversy exists as to the ideal first-aid method for rewarming the accidental hypothermia victim in the field. There is little objective data supporting past recommendations, which have unfortunately been based on pre-hospital anecdotal reports and personal experience from the hospital management of hypothermia (82-90). Some authors recommend active external rewarming (91-95) while others recommend active internal rewarming for the treatment of accidental hypothermia (96-99). Conflicting results comparing the degree of body core temperature afterdrop between active external and internal rewarming (96,97,100) may be explained by the differences in protocols for rewarming for human and animal model experimentation.

Most authors agree that rapid peripheral rewarming is the treatment of choice in rapid onset accidental hypothermia, such as cold-water near-drowning (91,93,100-104). However, rapid peripheral rewarming may lead to potential complications from peripheral vasodilation leading to ventricular fibrillation (34,101,105-106) and cardiac arrest (107) from sudden cooling of the myocardium as well as hypovolemic shock secondary to a reduced central blood volume (105-106). The risk of hypovolemic shock due to diuresis and fluid redistribution is especially severe in hypothermia of slow-onset (75). This has led to the recommendation that hypothermia of slow onset should be treated by slow rewarming whereas rapid rewarming is thought to be safe for hypothermia of rapid onset (106,108). Ventricular fibrillation and rewarming hypovolemic shock are potential problems that may go undetected and be difficult to manage in the remote setting without pre-hospital medical training, vital signs interpretation, and cardiac monitoring.

To avoid the risks of peripheral rewarming, core rewarming techniques have been proposed to deliver heat to the central organs and blood volume, thus avoiding peripheral vasodilation. Core rewarming methods found to be effective, but only useful in the hospital setting, include peritoneal lavage or dialysis using warm fluids (83), extracorporeal circulation rewarming (86,89), thoracotomy with warm fluids bathing the heart (109), endotracheal intubation with either warm air ventilation (110) or combined with warmed intravenous fluids (111). Unfortunately, these techniques cannot be safely accomplished in the field setting and are not available in advanced helicopter air ambulances. One new technique showing high promise in animal studies is radio-frequency (RF) rewarming (112). Recently, an RF vest was developed by the Naval Aerospace Medical Research Laboratory, Pensacola, FL and will soon be evaluated under human experimental trials.

In 1972, inhalation rewarming through voluntary inspiration of warm, humidified oxygen was first proposed by Lloyd et al. (113) as a first aid measure for central rewarming. Lloyd et al. (113-114) proposed that the clinical benefit of inhalation rewarming was due to the elimination of respiratory heat loss rather than additional heat supplied. However, this assumption was not proven from observations made from an unspecified number of hypothermic patients. Since then, many studies have been done which both support (96-97,100,115) and refute (98,116-117) the assumption that inhalation rewarming influences the rewarming rate by heat being delivered to the central body core. Differences in experimental protocol may explain the disparity of results and current confusion over inhalation rewarming strategies. Such differences include: various rewarming water temperatures (96-98,100),

inhalation temperatures (97,116) and room air temperatures (97) during rewarming, delays in rewarming possibly affecting afterdrop (98,100,115), varying amounts of insulation worn by a subject during inhalation rewarming (96-98,100), and uncomparable body core temperatures made at various sites.

Esophageal temperature, by measuring the central, thoracic temperature, is a useful temperature to determine the influence of inhalation rewarming on afterdrop and rewarming rate. However, in the three studies supporting inhalation rewarming, esophageal temperature was measured in only one subject (96,100) or in an unspecified number of patients (113-114). Recently, a study by Romet and Hoskin (117) on eight subjects demonstrated inhalation rewarming did not have any effect on the degree of esophageal temperature afterdrop or rewarming rate compared to shivering alone. However, this well controlled study evaluated both shivering and inhalation rewarming in room air (21°C). Rewarming in a colder environment, similar to the field situation, may have uncovered a benefit of inhalation rewarming to limit respiratory heat loss, thus minimizing afterdrop and accelerating the rewarming rate.

D. FIELD TREATMENT OF HYPOTHERMIA, CURRENT RESEARCH

A prospective, randomized, controlled study was recently conducted at the Navy Experimental Diving Unit on eight diver-subjects evaluating various field rewarming techniques and equipment (118). The purpose of the study was to determine the effect on afterdrop and rewarming rate of peripheral rewarming and inhalation of warm, humidified air. All eight diver-subjects wore dry suits with thin, long underwear insulation and were immersed to the neck in 0°C ice water, using one-man immersion tanks. The hands and feet were kept out of an ice water bath to avoid nonfreezing cold injury. Cooling was terminated when the rectal temperature reached 35°C, the limit of ethical human experimentation. No afterdrop occurred below 34°C during the rewarming phase. Removing the diver from the ice bath onto the rewarming stretcher was done with great care to prevent exercise induced afterdrop. The rewarming phase simulated field rescue conditions inside a tent in a cold, dry environment of 2°C. The experimental design compared two rescue sleeping bags with no peripheral rewarming; one rescue bag, cold vs. warmed by a charcoal heater/blower; one rescue bag, cold, with and without innalation rewarming; and one rescue bag, warmed, with and without inhalation rewarming. This allowed a statistical comparison of all techniques, separately or combined, with each diver-subject serving as his own control. The two rescue bags were the Thermo-Recovery Capsule (model LGS-TRC-A, Lifegaard Systems, Inc., Hurley, NY), and the Heat Pac Rescue Bag (A.B. Russell Co., Waitsfield, VT). The Heat Pac Rescue Bag was also heated with a charcoal burner and battery operated blower, also distributed by A. B. Russell. The warm air baffles surrounded the diver's chest, neck, and axilla. The neck and axilla are two areas of high heat exchange, ideal for peripheral rewarming. Inhalation rewarming was accomplished with humidified air warmed by either electrical or propane portable heaters to the highest tolerated inspiratory temperature (determined to be 45°C) and delivered by facemask (Heat Treat, Thermogenesis, Victoria, B.C., Canada). Esophageal and rectal core temperatures were recorded, on-line by a diver monitoring system, described elsewhere (4). As mentioned, the CorTemp pill unfortunately yielded inaccurate data and unreliable data collection, so gastrointestinal temperature could not be compared to esophageal temperature in this study.

Afterdrop was characterized by: (a) the relative and (b) absolute drop in both esophageal and rectal temperatures following removal from the ice bath, (c) the time until the maximum afterdrop and (d) time until the two core temperatures returned to the temperature when cooling was terminated. Rewarming rates were also determined for 30 and 60 mins following the maximum afterdrop. There were no statistically significant differences in the cooling rates in the ice bath, or the core temperatures at the beginning of rewarming.

Despite rigorous control of confounding variables, and highly reproducible results in all diver-subjects tested, there were no significant differences in afterdrop or rewarming rates by either esophageal or rectal temperatures for: rewarming by shivering in either bag (of equal loft and insulation); peripheral rewarming vs. shivering in a cold bag; inhalation rewarming with either cold or heated bags, and no difference between a combination of inhalation plus peripheral rewarming vs. a cold bag with no inhalation rewarming.

These negative results are not surprising when one considers that inhalation rewarming improves heat balance by only 23 kcal/hr (10 kcal/hr heat is delivered by inhalation rewarming plus preventing the loss of 13 kcal/hr from breathing cold air) (111). This amount of heat is very small considering it is only 1/3 of basal metabolic heat production and the human body can increase metabolic rate 5 times with vigorous shivering (119).

Both rescue bags proved to be too thin to be used alone. This was ideal for our purposes of evaluating the benefit of adding heat, peripherally or by inhalation, with the diver-subjects marginally insulated and breathing cold air as the control condition.

There are serious safety hazards with both of these peripheral and inhalation rewarming techniques that have not been reported. The Heat Pac charcoal burner/blower unit exhaust tube easily disconnects and is only 1-foot long. This causes a lethal risk of carbon monoxide (CO) poisoning due to a measured 500-1000 ppm CO exhaust from the unit (118). If the battery is placed in backwards, the fan spins in reverse, which will put out the combustion reaction, unknowingly.

The Heat Treat units that boil water to both humidify and heat the inspired air had numerous problems. Propane is difficult to light below -20 to 30°C (-4 to -22°F) due to low vapor pressure and moisture in propane that may freeze. Butane will never light if the outside temperature is below 4°C (40°F) due to a low vapor pressure. When lighting the propane unit, even in still air, there were frequent large ignitions which have burned the hair off the operator's hand and arm. The propane unit also overheated and melted the plastic and nylon it was standing on and was too hot to handle or carry. The CO output from the Heat Treat units was not measured, but due to the high combustion rates, it may be dangerously high inside a tent without adequate ventilation. The consumption of fresh water is very high, approximately 450 ml (15.2 fl. oz.) every hour. This puts great demand on having abundant, fresh water that must be very clean for this inhalation therapy. In the high latitudes or at sea, fresh water is at a premium and always needed for hydration. The collection of water from condensation in the breathing tubes created very uncomfortable negative pressure breathing with high respiratory rates seen in mild hypothermia. This was severe enough to increase the anxiety of one hypothermic subject to nearly terminate the experiment. The water going down

the nose, in the mouth, into the eyes and saturating clothing around the face, head and neck was disliked by all eight diver-subjects. It was also very difficult at first to maintain constant inspiratory temperatures between 43-45°C. If the temperature became uncomfortably hot, the subject could easily be burned in the face even if the breathing hose was immediately disconnected at the heater unit. The electrical unit did not allow the temperature to be controlled any easier, using the same mixing valve of steam to fresh air.

With a lack of significant physiological benefit, plus these safety hazards, peripheral and inhalation rewarming techniques cannot be recommended for the field treatment of hypothermia. From these studies, the best recommendation would be for removing all wet clothing or dive suits, and maximizing thermal insulation by using the Heat Pac Rescue Bag, which opens up easily to accept a thick sleeping bag on the inside. The hood covering, side straps for use as a litter and pulling straps in the front make it an ideal rescue bag if used with additional insulation. The Heat Pac Rescue Bag could be packed and stored with an Arctic-grade sleeping bag. High quality down sleeping bags can be water proofed and vacuum packed as small as the volume of a large textbook. Using a vapor barrier, or frost liner inside the sleeping bag will prevent loss of insulation from insensible water loss freezing inside the sleeping bag, especially from respiratory insensible water loss. A ground pad, inflatable or foam-type, will prevent conductive heat loss into the ground, too.

One internationally recognized authority in extreme cold weather clothing, equipment and training is Exploration Products, Spokane, WA. Telephone: (509) 927-8101 or (800) 448-7312. This organization has had numerous military contracts to outfit U.S. and Canadian Forces for Arctic deployment, including diving operations in the Arctic Sea.

III. DISCUSSION AND CONCLUSIONS

A. FIELD TREATMENT OF HYPOTHERMIA, FIRST AID MANAGEMENT

Maintain the patient in a horizontal position after removal from the water. As discussed, immersion hydrostatically increases the intrathoracic blood volume, which also improves cardiac output by as much as 60% (120). Hypotension may occur upon removing the victim from the water in the erect position. This also may precipitate lethal arrhythmias. Handle the hypothermic patient gently, to avoid stimulation of the circulation or heart, which may precipitate arrhythmias. Do not let someone who is suspected of being hypothermic, walk or exercise until they are fully evaluated. Exercise, especially after cold water immersion, is suspected to cause sudden death if the victim is moderately hypothermic. This may be due to profound afterdrop along with hypotension. Suspected hypothermic victims may need to be restricted to lying down due to the mental confusion and agitation that is commonly observed.

The primary survey should rely on the First Aid principle of A, B, C: Airway, Breathing and Circulation, but add D for degrees and disability. Consider both hypothermia for degrees and cervical spine injury for disability. Most authors agree that cardiopulmonary resuscitation (CPR) should not be withheld, due to a lack of evidence that CPR would provoke lethal arrhythmias (79). There is no evidence to recommend that the rate of CPR be reduced,

either, to support a reduced metabolic rate. Remember that severe hypothermia victims look dead. The rule, which may need to be enforced at the accident scene is "no one is dead, until they are warm and dead."

Remove all wet clothing, including wet suits and the dry suit undergarment material if it is even moist. It is untrue that wet wool or even synthetic material such as Thinsulate maintains most of the insulation if wet. Recent studies at the Navy Experimental Diving Unit demonstrated more than 88% of insulation is lost by moderate wetting of the best of synthetic undergarment materials (63-64). Also, radiant barrier material, "space blankets", did not reflect any significant energy (63-64), and would only serve to act as a wind barrier. Large trash bags are inexpensive and can be easily modified with adding a hole for the head to help prevent convective and evaporative heat loss for the victim in the field. As recommended above, wrap the patient in the thickest, dry insulation possible. Place the patient in an Arctic-grade sleeping bag inside the Heat Pac Rescue Bag or similar insulation of many blankets, with a foam pad beneath the victim to prevent conductive heat loss into the ground, deck of ship or ice. Cover the patient's head with thick hats, to prevent heat loss. Remember, the scalp blood flow is not well autoregulated like cutaneous blood flow and heat loss from the head is very great.

Protect the patient from wind chill, and build a fire if possible. The radiant barrier, space blanket can be used to not only act as a wind screen, it will reflect the high radiant energy from the fire back to the patient. Arrange for medical evacuation (MEDEVAC), and begin your physical exam as outlined above. If rectal core temperature monitoring is available, let it guide you, but rely on your clinical exam and constant vital sign monitoring to treat the patient.

For mild hypothermia, providing there are no underlying metabolic disorders, drug or alcohol ingestion which may limit the shivering response, spontaneous rewarming will safely occur if the patient is well insulated. The most common agreement for the treatment of moderate to severe hypothermia is for these patients to be highly insulated, remain in a "metabolic icebox" condition, and be actively rewarmed only in the well controlled conditions of a hospital familiar with invasive rewarming techniques, such as peritoneal dialysis, extracorporeal rewarming, intubated inhalation rewarming or open thoracotomy with lavage (13,78,121).

In the field, supportive care is directed to help the patient generate heat by providing hydration and if tolerated, nutrition. There is no physiological evidence against the use of peripheral rewarming techniques, such as hot water bottles, chemical heat packs or the Heat Pac charcoal burner unit around the head, neck, axilla and groin, inhalation rewarming, or even the questionable benefit of body-to-body contact inside a large enough sleeping bag. The safety hazards listed above should guide the rescuer on whether the risks and burden of certain expensive gear outweigh the unproven possible benefit. The lack of sound advice comes from a paucity of physiological data on the benefit of field rewarming techniques used on victims with moderate to severe hypothermia. Human studies can only be done on volunteers with rectal core temperatures reduced to only 35°F (95°F).

It has also been documented that chemical heat packs can cause third degree burns, with temperatures up to 77°C (170°F) (122). Hot showers, which are available on larger ships and on extended Arctic deployments, may feel good but suppress shivering (123) and may not effectively rewarm due to little heat transfer plus increased evaporative heat loss. Hot showers may also induce hypotension and syncope in hypothermic divers and, therefore, should be avoided. Vigorous rubbing of the extremities does not help to improve heat generated by the body. This may precipitate muscles to cramp, and stimulate cold venous blood back to the already irritated myocardium. Urination can lead to fainting, called micturition syncope. With blood pressure being labile, have the hypothermia patient urinate as close to horizontal body position as possible.

Intravenous (IV) fluids to combat the severe dehydration following cold water diving should be isotonic such as normal saline or lactated Ringers. Lactated Ringers should be avoided in severe hypothermia since the liver may not metabolize lactate well. The IV fluid should be prewarmed (pan of hot water or microwave, if available) up to 46°C (115°F) to prevent further cooling of the patient. If there is a protective gag reflex, warm, sugar containing fluids by mouth will not add that much warmth, but offer hydration, nutrition and psychological relief to the patient. There is no evidence that drinking warm fluids will be harmful by inducing any afterdrop. Alcohol and caffeine containing beverages should be avoided due to the unwanted diuresis. Warm electrolyte/sugar sports drinks are ideal in a field situation for oral hydration and moral support.

Bath rewarming, in the field, for moderate to severe hypothermia patients should be avoided. There should be no delays for the MEDEVAC of these patients to a hospital for complete medical monitoring during proven, invasive rewarming procedures outlined above. Hot tubs are becoming common near cold water dive sites, and portable units are easily constructed at remote Arctic dive sites. A diver with mild hypothermia will not tolerate sudden immersion in water above 30°C (86°F), due to the sensation of scalding pain, especially hands and feet (118). We recently demonstrated that rapidly raising the temperature from 30°C to 41.1°C (86 - 106°F) over 15 mins was easily tolerated by eight hypothermic divers (118). Compared to comfortably warm water (37°C, 99°F), the 41.1°C water temperature reduced the time to sweating and an acceptable rectal and esophageal core temperature by from 60 to 24 mins. Furthermore there were no differences between the core temperatures at the onset of sweating, which terminated rewarming, in bath temperatures from 37°C to 41.1°C (118). If a small hot tub is available, but the temperature cannot be adjusted, any tolerated temperature above thermoneutral, 35°C, would be recommended, with the skin splashed with warm water to lessen the pain of warm water immersion.

With difficulty in medically monitoring the patient while immersed, bath rewarming should not be used for other than stable patients with mild hypothermia. Regarding the risk of having the extremities in hot water, a recent study did not find any difference between leaving arms and legs out or in the bath, to influence the afterdrop or rewarming rate (124). Unfortunately, warm water immersion may induce arrhythmias from cold venous blood returning to the heart in patients colder than those used in this study. The benefit of warm water immersion to support circulation and increase the rewarming rate must be weighed against the possibility of inducing arrhythmias.

Arrhythmia prophylaxis with lidocaine or bretylium is an area under investigation now. Although frequently used, the doses have not yet been recommended based on a lack of physiological studies of dose response with varying temperatures of cooled myocardium. Until these studies are done, standard Advanced Cardiac Life Support (ACLS) doses are recommended. Caution should be applied with using any ACLS recommended drug with hypothermia reducing peripheral circulation. Rewarming can release high drug concentrations, leading to toxic reactions.

B. PREVENTION OF HYPOTHERMIA DURING DIVING

Poor dive planning, obligating a cold diver to remain at rest during prolonged decompression or repetitive dives with insufficient rewarming are usually the reasons for mild hypothermia during diving operations (60-62). Frequently, the coldest people conducting cold water diving operations are the tenders who must remain at rest, exposed to the cold while they support their divers. These tenders then rotate to become divers, but they are already cold, dehydrated and frequently long overdue for a meal. Dive supervisors must care for their tenders and insure adequate rewarming and rehydration before sending divers into the water. For reasons unclear, it seems to take over 24 hours to recover from even the mildest of hypothermia, after proper hydration, body core temperature and nutritional debts have been paid back. To reduce exposure to both topside support crew and divers, temporary dive shelters with portable heaters have been used with great success (60-62). Caution should be exercised with these heaters, since improperly adjusted, they put out dangerously high levels of carbon monoxide. This is not an uncommon emergency during cold water diving operations in the northern latitudes (60-62). Despite the best of dive plans, there will be accidents with lost divers, injuries underwater or lost hot water supply to the divers hot water suit or gas supply. Divers can also become very hypothermic following a dive, even in the warmer waters of the Gulf of Mexico. During a long, fast ride back into shore, the high wind chill with sea spray causes substantial heat loss from evaporation.

C. RESEARCH IN ACTIVE THERMAL PROTECTION FOR DIVING

In order to encourage international collaboration between various laboratories studying hypothermia in diving, the Navy Experimental Diving Unit and the Defense and Civil Institute of Environmental Medicine organized a recent workshop entitled, Diver Thermal Protection (125). Over 30 papers were presented from the United Kingdom, Norway, Canada and the United States from both commercial, scientific and military diving authorities. The workshop discussed currently used thermal protection diving gear, passive and active thermal protection research, physiological considerations and measurement techniques. Improved scientific exchange has continued as a result of this workshop and future workshops in thermal protection are strongly encouraged.

In the area of active thermal system (ATS) protection for the free-swimming diver, the Navy Experimental Diving Unit recently completed over 50 man-dives in 2°C water evaluating two, closed circuit tube suits (5). Experimental dives were conducted in a newly designed flume allowing unrestricted free-swimming and physiological monitoring in a constant current of either 2°C fresh water or -2°C sea water (126-127). The results, which are very encouraging, are based on physiological assessment of heat loss, core temperatures, skin temperatures, body and suit insulation and metabolism (5). These ATS tube suits are intended

to give active thermal protection for divers planning to be at rest in extreme cold water for many hours, and yet be flexible for free-swimming with a man-carried heat source. There is also on-going research in active hand thermal protection at the Naval Medical Research Institute and the Defense and Civil Institute of Environmental Medicine. The tube suit concept will not only assist military, commercial and scientific divers, it may have an application in delivering sufficient heat for the field management of moderate to severe hypothermia, someday.

REFERENCES

1. Francis T, Golden F. Non-freezing cold-injury: the pathogenesis. *Journal of Royal Naval Medical Service* 1985; 71:3-8.
2. Lockhart JM. Effects of body and hand cooling on complex manual performance. *J of Appl Physiol* 1966; 50(1):57-59.
3. Sterba JA. Portable diver physiological monitoring during Arctic diving operations. Navy Experimental Diving Unit (Panama City, FL) Report, 1990; in press.
4. Braun JR, Sterba JA. Diver monitoring systems, on-line and portable for thermal and metabolic measurements. *Undersea Biomed Res* 1989; Supplement to Vol. 16:49.
5. Sterba JA. Physiological evaluation of two actively heated tube suits during diving in 2°C water. Navy Experimental Diving Unit (Panama City, FL) Report, 1990; in press.
6. Francis T. Non-freezing cold injury: A historical review. *Journal of Royal Naval Medical Service* 1984; 70:134-139.
7. Sullivan BJ, LeBlanc MF. Effect of inositol and rapid rewarming on extent of tissue damage due to cold injury. *Am J Physiol* 1957; 189(3):501-503.
8. Fuhrman RA, Fuhrman GJ. The treatment of experimental frostbite by rapid thawing. *Medicine* 1957; 36:465-487.
9. Thalmann ED, Schedlich RS, Broome JR, Barker PE. Evaluation of passive protection systems for cold water diving. Institute of Naval Medicine (Alverstoke Hants, UK) Report 25/87, 1987.
10. Maclean D, Emslie-Smith D. Accidental Hypothermia. JB Lippincott Co., Philadelphia, PA. 1977.
11. Harnett RM, Pruitt JR, Sias FR. A review of the literature concerning resuscitation from hypothermia: Part I-The problem and general approaches. *Aviat Space Environ Med* 1983; 54(5):425-434.
12. Harnett RM, Pruitt JR, Sias FR. A review of the literature concerning resuscitation from hypothermia: Part II-Selected rewarming protocols. *Aviat Space Environ Med* 1983; 54(6):487-495.
13. Ferguson J, Epstein F, van de Leuw, J. Accidental Hypothermia. In: *Emergency Medicine Clinics of North America*. Philadelphia, PA: W. B. Saunders, 1983; 1(3): 619-637.
14. Mittleman KD, Mekjavic IB. Effect of occluded venous return on core temperature during cold water immersion. *J Appl Physiol* 1988; 65(6):2709-2713.

15. Vaughan WS Jr. Diver temperature and performance changes during long-duration, cold water exposure. *Undersea Biomed Res* 1975; 2(2):75-88.
16. Arborelius M, Jr, Balldin UI, Lilya B, Lundgren CEG. Haemodynamic changes in man during immersion with the head above water. *J Aerospace Med* 1972; 43(6):592-598.
17. Young AJ, Muza SR, Sawka MN, Pandolf KB. Human vascular fluid responses to cold stress are not altered by cold acclimation. *Undersea Biomed Res* 1987; 14:215-228.
18. Deuster PA, Smith DJ, Smoak BL, Singh A, Montgomery LC, Doubt TJ. Prolonged whole body cold water immersion: fluid and ion shifts. *J Appl Physiol* 1989; 66:34-41.
19. Deuster PA, Smith DJ, Smoak BL, Doubt TJ. Fluid and electrolyte changes during cold water immersion. *Undersea Biomed Res* 1987; 14(2):11-12.
20. Keatinge WR, Evans M. The respiratory and cardiovascular response to immersion in cold water and warm water. *Q J Physiol* 1961; 46:83-94.
21. Keatinge WR, Nadel JA. Immediate respiratory response to sudden cooling of the skin. *J Appl Physiol* 1965; 20:65-69.
22. Cooper KE, Martin S, Riben P. Respiratory and other responses in subjects immersed in cold water. *J Appl Physiol* 1976; 40:903-910.
23. Pozos RS. Cold stress and its effects on neural function. In: Laursen GA, Pozos RS, Hempel FG, eds. *Human Performance in the Cold*. Bethesda, MD: Undersea Medical Society, 1982:25-36.
24. Hayward MG, Keatinge WR. Progressive symptomless hypothermia in water: possible cause of diving accidents. *Br Med J* 1979; 1:1182.
25. Piantadosi CA, Thalmann ED, Spaur WH. Metabolic response to respiratory heat loss-induced core cooling. *J Appl Physiol* 1981; 50(4):829-834.
26. Pendergast DR, DeBold AJ, Pazik M, Hong SK. Effect of head-out immersion on plasma atrial natriuretic factor in man. *Proc Soc Exp Biol Med* 1987; 184:429-435.
27. Andrews I, Orkin LR. Environmental cold and man. *Anesthesiology* 1964; 25:549.
28. Coniam SW. Accidental Hypothermia. *Anesthesiology* 1979; 34:250.
29. Okada M, Nishimura F, Yoshino H, Kimsuru M, Takanori O. The J wave in accidental hypothermia. *J Electrocardiol* 1983; 16(1):23-28.

30. Osborn JJ. Experimental hypothermia: Respiratory and blood pH changes in relation to cardiac function. *Am J Physiol* 1953; 175:389.
31. Ree NJ. Electrocardiographic changes in accidental hypothermia. *Br Heart J* 1964; 26:566.
32. Emslie-Smith D, Sladden GE, Stirling GR. The significance of changes in the electrocardiogram in hypothermia. *Br Heart J* 1959; 21:343.
33. Lloyd EL. Factors affecting the onset of ventricular fibrillation in hypothermia. *Lancet* 1974; 2:1294.
34. Stine RJ. Accidental hypothermia. *JACEP* 1977; 6:413.
35. White JD. Cardiac arrest in hypothermia. *JAMA* 1980; 244:2262.
36. Siebke H, Rod T, Brievik H, Lind B. Survival after 40 minutes submersion without cerebral sequelae. *Lancet* 1975; June 7.
37. *Time Magazine* 1977; Aug 27:73-74.
38. U.S. Coast Guard. Cold water drowning. A new lease on life. Nov 1977; MIHCU-SG-77-104. CG-513.
39. Nemiroff MJ, Saltz GR, Weg JG. Survival after cold-water near-drowning; the protective effect of the diving reflex. *Am Rev Respir Dis* 1977; 115(Suppl):145.
40. Asmussen E, Kristiansson NG. The "Diving Bradycardia" in exercising man. *Acta Physiol Scand* 1968; 73:527-535.
41. Gooden BA, Lehman RG, Pym J. Role of the face in the cardiovascular responses to total immersion. *Aust J Exp Biol Med Sci* 1970; 48:687-690.
42. Hong SK, Moore TO, Seto G, Park HK, Hiatt WR, Bernauer EM. Lung volumes and apneic bradycardia in divers. *J Appl Physiol* 1970; 29:172-176.
43. Moore TD, Lin YC, Lally DA, Hong SK. Effects of temperature, immersion, and ambient pressure on human apneic bradycardia. *J Appl Physiol* 1972; 33:36-41.
44. Paulev PE. Respiratory and cardiovascular effects of breath-holding. *Acta Physiol Scand (Suppl)* 1969; 324:1-116.
45. Song SH, Lee WK, Chung YA, Hong SK. Mechanisms of apneic bradycardia in man. *J Appl Physiol* 1969; 27:323-327.
46. Stromme SB, Kerem D, Elsnor R. Diving bradycardia during rest and exercise and its relation to physical fitness. *J Appl Physiol* 1970; 28:614-621.

47. Arnold RW. Extremes in human breath-hold, facial immersion bradycardia. Undersea Biomed Res 1985; 12(2):183-190.
48. Sterba JA, Lundgren CEG. Influence of water temperature on breath-holding time in submerged man. Undersea Biomed Res 1979; 6(Suppl):29-30.
49. Hayward JS, Hay C, Mathews BR, Overwheel CH, Radford DD. Temperature effects on the human dive response in relation to cold-water near-drowning. J Appl Physiol 1984; 56:202-206.
50. Sterba JA, Lundgren CEG. Diving bradycardia and breath-holding time in man. Undersea Biomed Res 1985; 12(2):139-150.
51. Sterba JA, Lundgren CEG. Breath-hold duration in man and the diving response induced by face immersion. Undersea Biomed Res 1988; 15(5):361-375.
52. Blix AS, Krog J, Myhre HO. The effect of breathing on the cardiovascular adjustments induced by face immersion in man. Acta Physiol Scand 1971; 82:143-144.
53. Campbell LB, Gooden BA, Horowitz JD. Cardiovascular responses to partial and total immersion in man. J Physiol (Lond) 1969; 202:239-250.
54. Corriol J, Rohner JJ. Role de la Temperature de l'eau dans la bradycardie d'immersion de la face. Arch Sci Physiol Paris 1968; 22:265-274.
55. Kawakami Y, Natelson BH, DuBois AB. Cardiovascular effects of face immersion and factors affecting diving reflex in man. J Appl Physiol 1967; 25:964-970.
56. Kobayashi S, Ogawa T. Effect of water temperature on bradycardia during nonapneic facial immersion in man. Jpn J Physiol 1973; 23:613-624.
57. Bergman SA Jr, Campbell JK, Wildenthal K. "Diving reflex" in man: its relation to isometric and dynamic exercise. J Appl Physiol 1972; 33:27-31.
58. Angelone A, Coulter NA Jr. Heart rate response to held lung volume. J Appl Physiol 1965; 20:464-468.
59. Openshaw PJM, Woodrooff GMF. Effect of lung volume on the diving response in man. J Appl Physiol 1978; 45:783-785.
60. Rey L, ed. Arctic underwater operations. Medical and operational aspects of diving activities in Arctic conditions. London, SW IV 1DE: Graham and Trotman, 1985.
61. Jenkins WT. Polar operations manual. SO300-85-MAN-010. Washington, D.C.: Commander, Naval Sea Systems Command, 1988.

62. Sterba JA. Arctic cold weather medicine. Navy Experimental Diving Unit (Panama City, FL), Report, 1990. in press.
63. Sterba JA. Cold water thermal protection: A comparative study. Undersea Biomed Res 1989; 16(Suppl):58.
64. Sterba JA, Hanson RS, Stiglich JF. Diver passive thermal systems (PTS) evaluation: Undergarment insulation, compressibility and absorbency. Navy Experimental Diving Unit (Panama City, FL) Report 10-89, August 1989.
65. Kuehn LA, Ackles KN, Cole JD. Survival test of submersible life support systems. Aviat Space Environ Med 1977; 48(4):332-338.
66. Kuehn LA, Ackles KN. Thermal exposure limits for divers. In: Johnson CE, Nuckols ML, Clow PA, eds. Hyperbaric diving systems and thermal protection. American Society Mechanical Engineers, NY. 1978.
67. Livingstone SD, Grayson J, Frim J, Allen CL, Limmer RE. Effect of cold exposure on various sites of core temperature measurements. J Appl Physiol 1983; 54(4):1025-1031.
68. Cooper KE, Kenyon JR. A comparison of temperatures measured in the rectum, esophagus, and on the surface of the aorta during hypothermia in man. Br J Surg 1957; 44:616-619.
69. Hayward JS, Eckerson JD, Kemna D. Thermal and cardiovascular changes during three methods of resuscitation from mild hypothermia. Resuscitation 1984; 11:21-33.
70. Nadel ER, Horvath SM. Comparison of tympanic membrane and deep body temperatures in man. Life Sci 1970; 9:869-875.
71. Mekjavic IB, Morrison JB, Brengelmann GL. Construction and position verification of a thermocouple esophageal temperature probe. IEEE Trans Biomed Eng 1984; 31:486-488.
72. Reuler JB. Hypothermia: Pathophysiology, clinical settings and management. Ann Intern Med 1978; 89:519-527.
73. Paton, BC. Accidental Hypothermia. Pharmacol Ther 1983; 22:331-377.
74. Lonning PE, Skulberg A, Abyholm F. Accidental hypothermia: Review of the literature. Acta Anaesthesiol Scand 1986;30:601-613.
75. Harnett RM, O'Brien EM, Sias FR, Pruitt, JR. Initial treatment of profound accidental hypothermia. Aviat Space Environ Med 1980; 51:680-687.
76. Fitzgerald FT, Jessop C. Accidental Hypothermia: A report of 22 cases and review of the literature. Adv Intern Med 1982; 27:128-150.

77. Bangs CC. Hypothermia and frostbite. In: Emerg Medicine Clinics of North America. Philadelphia, PA: W.B. Saunders, 1983; 1(3): 619-637.
78. Zell SC, Kurtz KJ. Severe exposure hypothermia: A resuscitation protocol. Ann Emerg Med 1985; 14(4):339-345.
79. Danzl DF, Pozos RS, Auerbach, PS, et al. Multicenter hypothermia survey. Ann Emerg Med 1987; 16(9):1042-1055.
80. Advance trauma life support course. Student manual. American College of Surgeons, Chicago, IL. 1985
81. Sterba JA. Evaluation of an impulse noise producing underwater tool on hearing in divers. Navy Experimental Diving Unit (Panama City, FL) Report 5-87.
82. Wickerstrom P, Ruiz E, Lilja GP, et al. Accidental hypothermia: Core rewarming with partial bypass. Am J Surg 1976; 131:622-625.
83. Lash RF, Burdette JA, Ozdil T. Accidental profound hypothermia and barbiturate intoxication. A report of rapid "core" rewarming by peritoneal dialysis. JAMA 1967; 201:269-270.
84. Pickering BG, Bristow GK, Craig DB. Case history number 97: Core rewarming by peritoneal irrigation in accidental hypothermia with cardiac arrest. Anesth Analg 1966; 56:574-577.
85. Linton AL, Ledingham IM. Severe hypothermia with barbiturate intoxication. Lancet 1977; 1:24-26.
86. Truscott DG, Firor WB, Clein LJ. Accidental profound hypothermia. Successful resuscitation by core rewarming and assisted circulation. Arch Surg 1973; 106:216-218.
87. Towne WD, Geiss WP, Yanes HO, et al. Intractable ventricular fibrillation associated with profound accidental hypothermia - Successful treatment with partial cardiopulmonary bypass. N Engl J Med 1972; 287:1135-1136.
88. Fell RH, Gunning AJ, Bardhan KD, et al. Severe hypothermia as a result of barbiturate overdose complicated by cardiac arrest. Lancet 1968; 1: 392-394.
89. Davies DM, Millar EJ, Miller IA. Accidental hypothermia treated by extracorporeal blood-warming. Lancet 1967; 1:1036-1037.
90. Neesemann ME, Busch HM Jr, Gundersen AL, et al. Asystolic cardiac arrest in hypothermia. Wis Med J 1983; 82:19-20.
91. Freeman J, Griffith L, Pugh, CE. Hypothermia in mountain accidents. Inter Anes Clin 1969; 7:997-1007.

92. Hayward JS, Collis M, Eckerson JD. Thermographic evaluation of relative heat loss areas of man during cold water immersion. *Aerospace Med* 1973; 44:708-711.
93. Jessen K, Hagelsten JO. Search and rescue service in Denmark with special reference to accidental hypothermia. *Aerospace Med* 1972; 43:787-791.
94. Myers RA, Britten JS, Crowley RA. Hypothermia: Quantitative aspects of therapy. *JACEP* 1979; 8:523-527.
95. Zachary LJ, Kucan JO, Robson MC, et al. Accidental hypothermia treated with rapid rewarming by immersion. *Ann Plast Surg* 1982; 9:239-240.
96. Collis ML, Steinman AM, Chaney RD. Accidental hypothermia: An experimental study of practical rewarming methods. *Aviat Space Environ Med* 1977; 48:625-632.
97. Hayward JS, Steinman AM. Accidental hypothermia: An experimental study of inhalation rewarming. *Aviat Space Environ Med* 1975; 46:1236-1240.
98. Marcus P. Laboratory comparison of techniques for rewarming hypothermic casualties. *Aviat Space Environ Med* 1978; 49:692-697.
99. Miller JW, Danzl DF, Thomas DM. Urban accidental hypothermia: 125 cases. *Ann Emerg Med* 1980; 9:456-461.
100. Hayward JS, Eckerson JD, Kemna D. Thermal and cardiovascular changes during three methods of resuscitation from mild hypothermia. *Resuscitation* 1984; 11:21-33.
100. Beeson PB, McDermott W. *Textbook of Medicine* 1971, WB Saunders Co., Philadelphia.
101. Burton AC, Edholm OG. *Man in a Cold Environment* 1955, Edward Arnold (Pub) Ltd., London.
102. Department of the Navy Publication, *Cold Injury*. *Navy Medicine* 1970; 5052-29.
103. Fernandez JP, O'Rourke RA, Ewy GA. Rapid active external rewarming in accidental hypothermia. *JAMA* 1970; 21:153-156.
104. Webb, P. Rewarming after diving in cold water. *Aerospace Med* 1973; 44:1152-1157.
105. Alexander L. The treatment of shock from prolonged exposure to cold, especially in water. Combined Intelligence Objective Sub-committee, Item 24. Office of the Publication Board, Department of Commerce, Washington, D.C., Report No. 250.

106. Keatinge WR. Survival in Cold Water. Blackwell Scientific Publications, Oxford and Edinburgh, Great Britain, 1969.
107. Rankin AC, Rae AP. Cardiac arrhythmias during rewarming of patients with accidental hypothermia. Br Med J 1984; 289:874-7.
108. Undersea Medical Society Workshop. Thermal problems in diving. Report Number WS:12-1-74, 1974. Undersea Medical Society, Inc., Bethesda, MD.
109. Pugh LG. Accidental hypothermia in walkers, climbers, and campers: Report to the Medical Commission of Accident Prevention. Brit Med J 1966; 1:123-129.
110. Shanks CA, Sara CA. Temperature monitoring of the humidifier during treatment of hypothermia. Med J Aust 1972; 2:1351-1352.
111. Shanks CA, Marsh HM. Simple core rewarming in accidental hypothermia with heat infusion, endotracheal intubation and humidification. Brit J Anesth 1973; 45:522-525.
112. White DJ, Butterfield AB, Nucc RC, Johnson C. Rewarming in accidental hypothermia: Radio wave versus inhalation therapy. Ann Emerg Med 1987; 16:50-54.
113. Lloyd EL, Conliffe NA, Orgel H, Walker PN. Accidental hypothermia: an apparatus for central re-warming as a first aid measure. Scot Med J 1972; 17:83-91.
114. Lloyd EL. Accidental hypothermia treated by central rewarming through the airway. Br J Anaesth 1973; 45:41-47.
115. Morrison JB, Conn ML, Hayes PA.. Influence of respiratory heat transfer on thermogenesis and heat storage after cold immersion. Clin Sci 1982; 63:127-135.
116. Guild W. Rewarming via the airway (CBRW) for hypothermia in the field? Journal of Royal Naval Medical Service 1978; 64:186-193.
117. Romet TT, Hoskin RW. Temperature and metabolic responses to inhalation and bath rewarming protocols. Aviat Space Environ Med 1988; 59:630-634.
118. Sterba JA. Efficacy of inhalation and peripheral rewarming techniques to treat hypothermia in the field. Navy Experimental Diving Unit (Panama City, FL), Report, 1990, in press.
119. Webb P. Cold exposure, in Bennett PB, Elliott DH (eds): The Physiology and Medicine of Diving, ed 2. San Pedro, CA, Best Publishers Co, 1975.

120. Farhi LE, Linnarsson D. Cardiopulmonary readjustments during graded immersion in water at 35°C. *Resp Physiol* 1977; 30:35-50.
121. Samuelson T, Doolittle W, Hayward J, Mills W, Nemiroff M. Hypothermia and coldwater near drowning: Treatment Guidelines. *Alaska Med* 1982; 24(6):106-111.
122. Feldman KW, Morray JP, Schaller RT. Thermal injury caused by hot pack application in hypothermic children. *Am J Emerg Med* 1985; 3(1):38-41.
123. Laursen GA, Pozos RS. Human performance in the cold; The challenge. In: Laursen GA, Pozos RS, Hempel FG, eds. *Human Performance in the Cold*. Bethesda, MD: Undersea Medical Society, 1982:1-11.
124. Hoskin, RW, Melinyshyn MJ, Romet TT, Goode RC. Bath rewarming from immersion hypothermia. *J Appl Physiol* 1986; 61(4):1518-1522.
125. Romet TT, Sterba JA, Nishi, RY, eds. Diver thermal protection. Undersea and Hyperbaric Medical Society Workshop, January 31- February 2, 1989, in preparation.
126. Braun JR, Sterba JA. Cold water swimming flume: Design and operation. *Undersea Biomed Res* 1989; 16(Suppl):49-50.
127. Braun JK, Sterba JA. Design, fabrication, and operation of a cold-water free-swimming flume for diving research. Navy Experimental Diving Unit (Panama City, FL) Report, 1990, in press.